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FIG. 1.—EARLY STAGE OF DISEASE.



FIG. 2.—LATER STAGE OF DECAY.

TRUNKS OF YOUNG WHITE ASH TREES, SHOWING EARLY AND LATER STAGES OF DISEASE.

U. S. DEPARTMENT OF AGRICULTURE.

BUREAU OF PLANT INDUSTRY—BULLETIN No. 32.

B. T. GALLOWAY, *Chief of Bureau.*

A DISEASE OF THE WHITE ASH CAUSED BY POLYPORUS FRAXINOPHILUS.

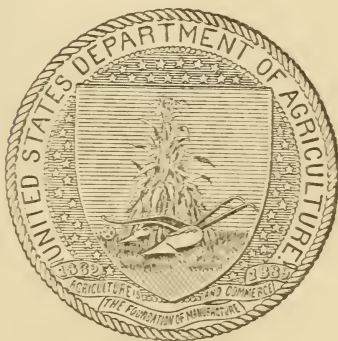
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VEGETABLE PATHOLOGICAL AND PHYSIOLOGICAL
INVESTIGATIONS.

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U. S. DEPARTMENT OF AGRICULTURE.

BUREAU OF PLANT INDUSTRY.

OFFICE OF THE CHIEF.

Washington, D. C., October 21, 1902.

SIR: I have the honor to transmit herewith, and to recommend for publication as Bulletin No. 32 of the series of this Bureau, the accompanying technical paper entitled "A Disease of the White Ash Caused by *Polyporus Fraxinophilus*."

This paper was prepared by Dr. Hermann von Schrenk, Special Agent in Charge of the Mississippi Valley Laboratory, Vegetable Pathological and Physiological Investigations, and it has been submitted by the Pathologist and Physiologist with a view to publication.

Respectfully,

B. T. GALLOWAY,

Chief of Bureau.

HON. JAMES WILSON,

Secretary of Agriculture.

PREFACE.

The accompanying paper treats of a disease of the white ash caused by *Polyporus fraxinophilus*, concerning which a number of inquiries have lately been made. It has been carefully studied by Dr. Hermann von Schrenk, who has charge of the Mississippi Valley Laboratory of Vegetable Pathological and Physiological Investigations, located at St. Louis. This disease is prevalent in the Mississippi Valley, which is the western limit of the white ash, and is particularly severe in Missouri, Nebraska, and eastern Kansas, fully 90 per cent of the trees in some localities being affected. The ash is extensively grown in parks and grounds, where the white rot does considerable damage. Its mode of growth and entrance into the tree may be taken as a type for many wound parasites destroying ornamental and shade trees, and it is believed that a knowledge of its life history and the methods to be used for combating it will prove of considerable benefit at this time both to foresters and others interested in the preservation of trees.

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OFFICE OF THE PATHOLOGIST AND PHYSIOLOGIST,
Washington, D. C., October 17, 1902.



CONTENTS.

	Page.
Introduction	9
White rot.....	9
Geographical distribution.....	10
Susceptibility to this disease	11
Method of attack	11
Description of diseased wood.....	11
The sporophore	12
Microscopic changes in the wood.....	14
Growth of the fungus in dead wood	17
Remedies.....	18
Description of plates	20

ILLUSTRATIONS.

PLATES.

	Page.
PLATE I. Sections of living white ash trees attacked by <i>Polyporus fraxinophilus</i> . Fig. 1.—Early stage of disease. Fig. 2.—Later stage of decay. Frontispiece.	
II. Fruiting bodies of <i>Polyporus fraxinophilus</i> on white ash. Fig. 1.— Fruiting body of <i>Polyporus fraxinophilus</i> . Fig. 2.—Two young sporophores on living ash. Fig. 3.—An old sporophore on living ash.....	20
III. Fig. 1.—Transection of healthy ash wood, stained with iodine. Fig. 2.—Transection of diseased ash wood, not stained.....	20
IV. Disease caused by <i>Polyporus fraxinophilus</i> . 1. Transection of ash wood, showing change in wood cells caused by fungus hyphæ. 2. Transection of medullary ray from brown wood layer, showing how the cells become filled with a brown humus compound. 3. A medullary ray, showing later stage of fungus attack. 4, 5. Tran- section of wood cells, showing various stages of change of wood into a brown humus compound. 6. Starch grains from medullary ray cell. 7. Starch grains from diseased wood. 8. Transection of rotted wood.....	20
V. Cross section of diseased trunk of white ash kept in a moist place for several weeks.....	20

TEXT FIGURE.

FIG. 1. Map showing distribution of <i>Fraxinus americana</i>	10
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A DISEASE OF THE WHITE ASH CAUSED BY POLYPORUS FRAXINOPHILUS.

INTRODUCTION.

The white ash is attacked by a number of fungus parasites, which grow on the living leaves and do more or less injury. *Accidium fraxini* Sch., the orange rust, is perhaps the one best known, as it grows on almost all species of ash, even the introduced forms. It occurs with varying frequency in successive years, and, so far as known, has appeared in epidemic form but once (1885). Among the fungi which grow as parasites on leaves are several species of *Gloeosporium* and *Sphaeropsis*, as well as *Septoria fraxini* and *Phyllosticta fraxini* Ell. & Mart. *Sphaeromena spina* Berk. & Rav. grows on young twigs, and kills a good many now and then.

The fungi mentioned above, to which several others might be added, rarely appear in sufficient numbers to do very much harm to the trees affected.

WHITE ROT.

There is one fungus, *Polyporus fraxinophilus* Pk., which grows in the heartwood of the trunk and branches of the white ash. This fungus changes the hard wood of the ash into a soft, pulpy, yellowish mass (Pl. I), making it unfit for lumber purposes. Diseased trees are ultimately blown down by windstorms. In regions where this disease is common the ash never grows to be a very large or very old tree. During the last year numerous inquiries have been made as to the causes of the white rot and how it could be prevented. In Forest Park, St. Louis, nearly all the white ash trees were diseased, and many were blown over by the wind.

A diseased tree is readily recognized by the large, conspicuously colored sporophores, which usually occur in considerable numbers, one or more at every branch stub. *Polyporus fraxinophilus* has been studied by the writer, particularly in Missouri, where it occurs in great numbers on the ash. It has been found elsewhere in the United States and has been reported from as far east as Albany County, N. Y.^a

^a Peck, C. H. Thirty-fifth Report, New York State Museum.

GEOGRAPHICAL DISTRIBUTION.

The distribution of this fungus is very interesting when considered with reference to its host. The white ash, as indicated on the accompanying map (fig. 1), is found throughout the entire eastern United States, growing as far westward as eastern Kansas and Nebraska. Judging from the very meager data now at hand, it seems that *Polyporus fraxinophilus* is most common near the western limit of the distribution of the white ash. It is very common in parts of Missouri, Kansas, Indian Territory, and Iowa. In the eastern United States, so far as the writer was able to ascertain, it is comparatively rare.

Near its western limit *Fraxinus americana* is at best a tree of medium size and development. On the dry limestone hills west of the Missis-

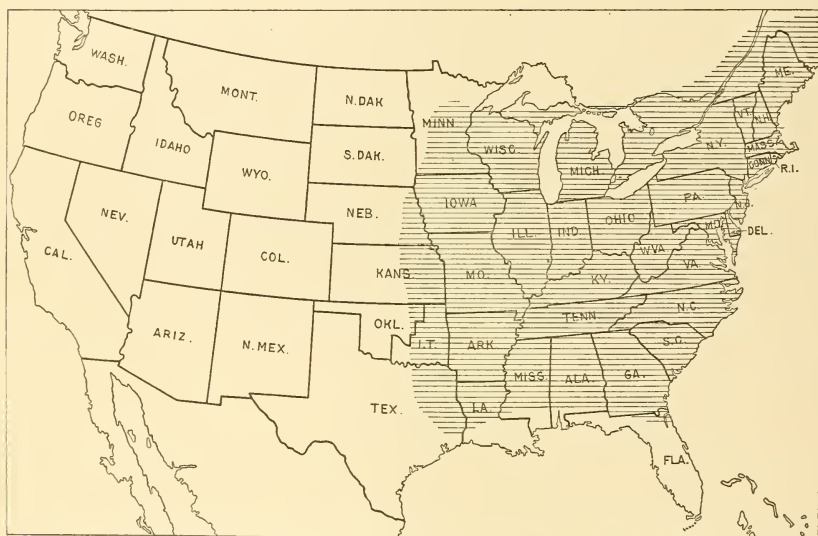


FIG. 1.—Map showing distribution of *Fraxinus americana* L.

sippi it grows slowly, as is evident from the sections shown on Pl. I, which are three-fourths natural size. In this region *Polyporus fraxinophilus* will be found on 90 per cent of the standing trees. The diseased trees were counted in two circumscribed localities, in neither of which was a tree more than 5 inches in diameter found to be sound.

The fact that in a given locality so high a percentage of the individuals of a species are diseased at a relatively early age may be explained by the greater virulence of the disease-causing factor or by the greater susceptibility of the individual; in this case, probably the latter. That this disease does not directly affect the living parts of the tree has no weight, for in the long run it affects it indirectly by undermining its support.

SUSCEPTIBILITY TO THIS DISEASE.

The question of the relative susceptibility of individual plants to a disease is a most interesting and at the same time a most obscure and difficult one to discuss. In the present instance it would seem that there might be some relation between the greater susceptibility on the part of the ash near its western limit and its generally weaker development at this limit. It will be an interesting point to determine, for instance, whether the rate with which branch wounds or stubs heal in Ohio and Pennsylvania is greater than in Missouri and Kansas. That the rate of growth is slower in the Western States we know.

Polyporus fraxinophilus has been reported as growing on living trees of *Fraxinus viridis* in Rooks County, Kans.^a

METHOD OF ATTACK.

Polyporus fraxinophilus attacks ash trees of all ages, usually, however, those more than 7 inches in diameter. The fungus begins its growth in a wound, or more often in a dead branch. It would perhaps be more correct to say that the fungus gains entrance into the tree at the point where the callus touches the branch stub. The branches of the ash are usually inclined upward at a considerable angle, and the callus leaves a groove between its outer surface and the branch stub in which water can collect. From sections of old branch stubs it appears that the earliest signs of fungus action are found in the outer parts of the dead stub close to this groove. The fungus grows down toward the center of the tree in the outer layers, and from there spreads to the main trunk up and down and laterally. It is quite usual to find a tree infected at two or more separate points. In a region where the sporophores are common and where each tree has many dead branches this is not at all surprising.

DESCRIPTION OF DISEASED WOOD.

The wood of the ash is uniformly straw yellow in color and shows little difference in tint between heart and sapwood. A gradual darkening of the wood near the center of the tree is the first indication of the presence of the fungus mycelium (Pl. I). In an irregular patch the wood looks as if stained, at first a very light brown, later on a darker brown. The broad bands of summer wood show this change in color most conspicuously. The next stage in the disease is marked by a bleaching of the color in the spring duct layers; these gradually turn back to the original straw color and then turn white in spots. The white color becomes more marked until the entire spring wood is white. It has a disintegrated appearance by this time, and shortly afterwards all the fibers fall apart. The dense bands of summer wood

^aEllis & Everhart. N. A. Fungi, No. 3302.

change more slowly. This gives rise to a banded appearance near the edge of the diseased area, more pronounced in some places than in others (see the lower part of Pl. I, fig. 2). Ultimately the whole wood ring turns into a loosely connected mass of fibers.

When the tree is first attacked it appears as if the changes described take place simultaneously over a large area (3 square inches in the tree shown in Pl. I, fig. 2), and that thereafter the change from sound to decayed wood goes on more slowly. This is the case in diseases of other trees, and is possibly accounted for by the fact that at first no products of metabolism interfere with the growth of the fungus, while later on these may retard growth to some extent.

The completely rotted wood is straw colored, very soft and nonresistent, and readily absorbs water. The disintegrating changes are by no means uniform, as a glance at Pl. I will show. The diseased areas have very irregular shapes; sometimes they involve the whole trunk, at other times only one side, depending somewhat on the point of infection and the shape of the trunk. In the trunk shown in the lower figure on Pl. I the fungus was growing in the seventh ring from the bark.

THE SPOROPHORE.

The sporophores of *Polyporus fraxinophilus* appear around the base of branch stubs, or in wounds, very soon after the original infection (Pl. II). With some trees—for instance, *Pinus echinata* attacked by *Trametes pini*—it appears that a good deal of wood is destroyed before any fruiting bodies of the fungus form. With the ash, fruiting bodies make their appearance when the wood shows signs of having decayed only a very short distance from the point of infection. In one tree, where the sporophore was developing at a branch stub, the heartwood was actually rotted for a distance of only 4 inches on either side of the base of the branch, while the characteristic discoloration extended for a foot in both directions from the stub. When the dead branch is a large one, small white knobs grow out at several points near its base (Pl. II, fig. 2), often as many as ten or a dozen. These knobs are almost white, very smooth, and adapt themselves to the irregularities of the rough bark. When the branch extends out horizontally, the sporophore frequently appears to be hanging from the under side of the branch (Pl. II, fig. 1). As the sporophores grow older they extend downward on the bark; in other words, become decurrent behind.

The mature sporophore is nearly triangular in cross section. Although fairly regular in form, there are many sporophores which are compound, i. e., composed of several superincumbent shelves or several shelves joined laterally. It has a broad rounded edge, which at first is white and gradually turns darker until it becomes somewhat straw colored. The older portions of the upper surface are dark brown or black, and are very hard and woody.

The youngest part grows out over the older portions, which makes old sporophores look somewhat sulcate. The main body of the mature sporophore is very hard and woody. It is obscurely zoned and pale brown or rust color. The pores are very regularly stratose. They are short and of regular cross section. The youngest ones are white, the older ones red brown. They extend from the point where the sporophore touches the bark almost to the edge of the sporophore.

There is some question as to what name ought to be given to this fungus. Two species of *Polyporus* growing on the ash have been described—*Polyporus fraxineus* (Bull.) Fr. and *Polyporus fraxinophilus* Pk. The European fungus is described by Bulliard^a and Fries^b as sessile, corky-woody, azonate, at first smooth, then concentrically sulcate, at first white, then red brown or brown, pale inside, pores minute, short, at first white, then red brown or rust color. This description accords fairly well with the specimens distributed in Thümen's Myc. Univ., No. 806, except that these specimens can hardly be called "woody." In 1881 Professor Peck described a fungus, *Polyporus fraxinophilus*, growing on ash trees in Albany County, N. Y., as follows:^c

Pileus sessile, thick, corky, subtriquetrous, narrow, somewhat decurrent behind, the first year whitish, with a minute whitish tomentum or hairiness, then gray, finally blackish, in old specimens concentrically sulcate, rimose, the substance within obscurely zoned, at first whitish, then isabelline or pale tawny, the margin obtuse; pores stratose, plane or subconvex, small, nearly equal, subrotund, the dissepiments obtuse, entire, whitish; spores white, broadly elliptical, .0003-.00035 inch long, .00025-.0003 inch broad. Pileus 2-4 inches long, 1-1.5 inches broad.

A comparison of the two descriptions will show that they are almost the same, differing in small details. Anyone who has tried to separate the species of this variable genus will have become impressed with the inadequacy of many of the older descriptions, and in the present instance it becomes a matter of extreme difficulty to determine whether the descriptions of Bulliard and Fries fit the American fungus. In most respects the latter agrees with the descriptions, except in the red-brown pores. The European specimens seen have red-brown pores. On the other hand, there can be no doubt as to the identity of the ash fungus with *Polyporus fraxinophilus* Pk. The decurrent pileus, at first with a whitish tomentum, later gray, and finally black, can not be mistaken for any other. In view of the fact that the only European specimens of *Polyporus fraxineus* available do not agree with the present fungus it is deemed best to retain the name given by Professor Peck for the present. It may be found necessary to make it a synonym of *Polyporus fraxineus* after a further comparison with European material.

^a Bulliard, M. Hist. des Champignons de la France, 1: 341, 1741.

^b Fries, Elias. Systema Myc., 1: 421; Rabenhorst's Kryptogamenflora, 1: 421, 1884.

^c Peck, C. H. Thirty-Fifth Report, New York State Museum, 1881, p. 136.

The fungus under discussion is one of the most distinct forms of the *Fomes* type of *Polyporus*, and considering the great variability of form of many species of this genus it can be said to be remarkably constant in most of its characters.

MICROSCOPIC CHANGES IN THE WOOD.

The minute changes which the wood cells undergo are marked by great distinctness and regularity. The wood of the ash forming the bulk of the trunk serves as a repository for large quantities of starch. Even in trees which are 75 to 100 years old one will find starch almost at the center. In the ash the starch occurs in the form of small grains (Pl. IV. fig. 6), filling the cells of the medullary rays and wood parenchyma. Fig. 1, Pl. III, represents a cross section of wood (cut in March), stained with iodine. The medullary rays appear almost as black lines.

One of the first changes noticeable in the wood when attacked by the ash fungus is in connection with this starch. The region where the starch changes is just outside of the dark line seen in Pl. I. The large grains (Pl. IV, fig. 6) appear to break up into numerous smaller ones (Pl. IV, fig. 7), and finally even these disappear. The change is a very rapid one, and transition stages are very rare. No such regular gradual dissolution of the grains occurs as is described by Hartig as taking place in oak wood attacked by *Polyporus sulphureus* and *Polyporus igniarius*. When stained with iodine one finds large grains now and then, with channels through them (Pl. IV, fig. 6), or more frequently some which look as if the center had been dissolved out. In several instances grains were found which stained brown with iodine at the edges. This brown color then gradually passed in toward the center of the grain.

No hyphæ are present in the wood where the starch is breaking up. This would indicate that a diastatic enzyme given off by the mycelium precedes the latter for some distance. The first hyphæ are generally several rings farther toward the middle of the trunk. The even extent of the solution strengthens this supposition, for in a limited area of one wood ring one and the same stage of dissolution is found at about the same distance from the point where the fungus begins its growth. After the disappearance of the smallest grains the cells formerly filled with starch appear empty for several cell rows inward. Shortly after the disappearance of the starch they become filled with a bright-colored substance, which is probably liquid at first and hardens after infiltration into the cells (Pl. IV, fig. 2). This substance, which is very soluble in alkalis, is probably some humus compound which must be regarded as a decomposition product. It is distributed throughout the medullary rays and the woody parenchyma, occupying almost the identical cells which had harbored the starch. This will

readily be comprehended by a comparison of Pl. III, figs. 1 and 2. Fig. 2 is from a photograph of an unstained section taken from the region of brown wood at the outermost edge.

It is rather difficult to determine the origin of this decomposition product. It is possibly the last product of a change in the starch grains, possibly also a substance derived from wood cells farther inward, which infiltrates into the medullary ray cells and wood parenchyma in advance of the fungus hyphæ. The latter is the probable explanation, for one finds the humus compound in the summer wood cells, which had very little starch originally. The humus compound appears to form in many of the wood cells, however, as a product of the walls. Figs. 4 and 5 of Pl. IV show various stages of this change. The cells *a* are sound wood cells, which have very thick walls and a very small lumen. The walls of cells marked *b* are very much thinner, and at these points they are coated with the humus compound. Such walls when stained with phloroglucin show no very sharp dividing line between the yellow humus compound and the apparently sound lignified wall. Cell *c* is completely filled with the humus mass. This evidence that the wall actually changes into the yellow mass is not very conclusive. The humus compound does not seem to be formed from the walls of the medullary ray cells, where it is found ultimately, for no signs of change are evident in the walls of these cells. The localized distribution of the humus substance is very striking. It is always absent from the wood cells of the spring wood (Pl. III, fig. 2) and from the large vessels. In the cells it appears to be as a solid mass, sometimes completely filling the lumen (Pl. IV, figs. 2 and 5), or in globules or plates adhering to the walls (Pl. IV, fig. 2). It is this substance which gives the brown color to the early stage of diseased wood.

The next stage in the dissolution of the wood cells takes place abruptly, and is rapid after it has once set in. The hyphæ of the fungus first evident in the medullary rays spread through the wood of both the spring and summer bands, branching in all directions. They give off an enzyme which attacks the inner parts of the wood cells, extracting the lignin. A transverse section of wood in this stage (Pl. IV, fig. 1) stained with phloroglucin presents a most striking picture. Here and there, in irregular groups and in all stages, one finds wood cells from which the hadromal has been removed; the extracted parts remain white and stand out in sharp contrast to the unaffected parts of the walls. In the figure the unaffected parts are shaded. The white parts represent delignified walls. The middle lamella is dissolved last and then the individual cells fall apart. When this takes place throughout larger areas, for instance, one or more wood rings become separated from one another, and this gives rise to the plates spoken of above. The white areas which are evident in the figures on Pl. I represent wood thus destroyed. The individual fibers

remain intact for some time, and are then gradually dissolved. In the oldest parts of diseased wood they are no longer present.

Wood partially destroyed in the manner just mentioned was stained with potassium permanganate. HCl and NH_4OH , according to the method recently described by Maule.^a

A dilute solution of the permanganate is allowed to act on the wood for a minute. The wood is then treated with strong HCl until no color is visible. A drop of ammonia is then added. The lignified walls stain a deep red, which in many respects defines the various parts of the walls more sharply than the phloroglucin reaction. The parts (Pl. IV, fig. 1), which do not stain with phloroglucin do not stain with the permanganate. The contrasting color between the lignified and delignified parts is even sharper. Maule claims that the permanganate reacted with an ether compound in the walls even after the removal of Czapek's hadromal. In the "delignified" wood cells of the ash even this compound (if there be a separate compound which reacts with the permanganate) is therefore absent.

In the ash wood the white fibers are not pure cellulose. The same is true of many similar fibers from oak wood destroyed by species of *Hydnum*, or *Polyporus igniarius*, and probably of other white fibers resulting from fungus action on wood. With chloriodide of zinc, the best cellulose reagent we have, these fibers stain a yellow brown, not blue. This would indicate that the change in the wall is not the same as in many of the conifers, where the so-called lignin is destroyed, leaving a comparatively pure cellulose, as determined by staining reaction and macrochemical analysis. This subject is simply referred to in this connection, as it will form the subject of a separate paper.

The change to an impure cellulose takes place locally, and generally very early in the course of the destructive action of the fungus. The mass of wood destroyed changes somewhat differently. The first changes noticeable are in the medullary rays and immediately adjoining cells. Very fine fungus hyphæ invade these cells, and shortly after the middle lamellæ disappear. Small cavities occur in thicker parts of this layer, i. e., where several cells touch (Pl. IV, fig. 3, *c*), and these increase in size (*v*), spreading laterally, until two or more join. Ultimately the individual cells become entirely isolated. The wood cells proper are gradually destroyed from within outward, the middle lamellæ remaining longest. The change from perfectly sound wood to wood entirely dissolved is a very abrupt one (Pl. IV, fig. 8). The hyphæ invade a cell and dissolve the wall. So rapid is this that no intermediate changes can be found. A piece of completely rotted wood, such as occurs in the center of a diseased trunk (Pl. I), is represented in Pl. IV, fig. 8. A more resistant piece of summer wood is

^aMaule, C. Das Verhalten verholzter Zellmembranen gegen Kalium permanganat, eine Holzreaction neuer Art. (Beiträge zur wissenschaftlichen Botanik, Vol. IV. Stuttgart, 1901.) (Reviewed in Bot. Cent., 89. 328, 1902.)

shown at one side. It is surrounded by an intricate mass of hyphæ, in which pieces of undissolved wood are held in much the relative position which they occupied in the sound wood. It will be seen that the wood is practically destroyed entirely. The mass of fungus hyphæ gives a soft, leathery, yielding consistency to the rotted material.

The young hyphæ are exceedingly fine, so much so that it requires a strong immersion lens to detect them. They are perfectly colorless, and remain so when older. Clamp connection occurs frequently.

GROWTH OF THE FUNGUS IN DEAD WOOD.

The mycelium of the fungus grows only in living trunks, so far as could be ascertained. It will grow out from infected wood when the latter is kept in a moist place, but only to a very small extent. A number of pieces of diseased ash trunks, each about a foot long, were placed in the mushroom cellar of the Missouri Botanical Garden, some with the cut surface in contact with the soil, others exposed to the moist air. In order to test whether dead wood could be infected, several healthy pieces of ash trunks, recently cut and of about the same diameter as the diseased pieces, were placed in contact with the smoothed end surfaces of the diseased pieces. After two or three days the hyphæ in nearly all the pieces began to grow out from the diseased areas (Pl. V), both from the brown areas and from the parts entirely decayed. This indicates that the fungus is equally active all through the diseased parts. In the pieces where the cut surfaces were exposed to the moist soil or air the hyphæ grew for some weeks, making a thick, tough felt. They gradually ceased growing after about three weeks. The sound ash trunks were firmly united to the diseased ones after three days, and after a week the fungus had so thoroughly united the two pieces that they could not be pulled apart, using a moderate amount of force. After three months the healthy pieces were examined. The hyphæ of the fungus had grown into the wood for a very short distance only. They had effected practically no change. A hard cushion of mycelium had formed between the two pieces, and this was turning brown and had evidently ceased growing. These tests show that under the conditions of temperature and moisture which permit of vigorous growth of several of the wood-destroying fungi growing on dead wood the mycelium of the ash fungus will not grow for any length of time. The sound wood placed in contact with the diseased wood was full of starch at the time, so it could not have been lack of food which prevented the growth of the hyphæ. A piece was removed from a sporophore immediately after it was brought in from the woods. The sporophore remained attached to a section of the trunk about a foot long. For several weeks hyphæ grew out from the injured surface, making a new rounded edge, doing so almost as rapidly as in the natural state.

REMEDIES.

The white ash is becoming more valuable as a lumber tree, and it is being grown extensively as an ornamental tree in parks and grounds. In limited areas it will pay to adopt measures which will tend to prevent the disease described in the foregoing pages, or at least to recognize diseased trees and use them for lumber, so as to save the parts still sound. A disease such as the white rot of the ash is a difficult one to combat after a tree is once badly diseased, for the fungus grows in the interior of the trunk, where it can not be reached. Trees which grow in forest tracts should be cut down when badly diseased, so as to prevent the spread of fungus spores. That a persistent cutting out of diseased trees will in a comparatively short period reduce the number of newly infected trees has been demonstrated repeatedly in European forests, where it is now often impossible to find many well-known forms of disease which were formerly comparatively common.

In parks and grounds diseased trees, when they appear healthy otherwise, need not necessarily be cut down, for the trees may remain alive and vigorous even when the heartwood is partially decayed. The only danger is that trees weakened in that way are liable to be broken off by windstorms. A diseased tree can be recognized as soon as the white punks or sporophores appear at a knot hole. As soon as a punk appears it can be cut out, and some of the diseased wood with it. The hole should then be filled with tar oil and left open for a time. Tar oil should be added from time to time, as a good deal will soak into the decayed wood, and thereby arrest the further growth of the fungus to some extent. If the hole made by removing the punk is a large one it should be covered with tar paper, so that no opening is left for water or dust to enter.

A sure method of combating this disease is by a careful system of pruning and the coating of all wounds with an antiseptic substance. Vigorously growing ash trees heal wounds rapidly, and after three or four years any ordinary-sized wound will be completely occluded. In treating trees planted in parks or gardens the pruning had best be done in the winter. Care should be taken to cut all branches as close to the trunk as possible, and after trimming the ragged edges of a cut the whole surface should be coated. Ordinary gas tar is the best substance for this purpose. If too hard it should be heated so as to be fairly liquid and then applied with a brush. The gas tar, especially when warm, penetrates for a considerable distance into the wood and prevents the development of the ash fungus. It forms an air-tight and water-tight cover which is not destroyed by weathering, and which at the same time is objectionable to insects.

Where the coating of wounds is carried on with care it will be entirely practicable and possible to prevent this ash disease.

PLATES.

DESCRIPTION OF PLATES.

PLATE I. (Frontispiece.) Sections of living white ash trees (*Fraxinus americana*) attacked by *Polyporus fraxinophilus* Pk. The upper figure shows an early stage; the lower, a later stage of the decaying process.

PLATE II. Fig. 1.—Fruiting body of *Polyporus fraxinophilus* Pk. growing out from a dead branch. This is a rather exceptional form of sporophore, which is found only on branches. Fig. 2.—Two young sporophores of *Polyporus fraxinophilus* Pk. growing on living ash. Fig. 3.—An old sporophore of *Polyporus fraxinophilus* Pk. growing on living ash.

PLATE III. Fig. 1.—Transection of healthy ash wood, stained with iodine so as to show the distribution of starch in the medullary ray cells and in the wood parenchyma surrounding the large ducts. This section is made just outside the dark line dividing sound from diseased wood (see Pl. I). Fig. 2.—Transection of diseased ash wood, not stained, showing the distribution of a humus compound in the medullary ray cells and in the wood parenchyma surrounding the large ducts. This section is made just inside the dark line dividing sound from diseased wood (see Pl. I).

PLATE IV. 1.—Transection of ash wood, showing one form of change in the wood cells caused by the fungus hyphæ. The darkly shaded parts are sound wood cells. The white parts are wood parts which do not stain with phloroglucin. (Magnification same as for fig. 2.) 2.—Transection of medullary ray from the brown wood layer, showing how the cells become filled with a brown humus compound, here shown by the dotted areas. In two cells the dry compound has cracked. 3.—A medullary ray, showing a later stage of fungus attack. The middle lamellæ are dissolved out, separating the individual cells from one another. Note the absence of the humus compound. (Magnification same as for fig. 2.) 4 and 5.—Transection of wood cells (highly magnified), showing various stages of change of wood into a brown humus compound. Note the great thickness of walls of neighboring sound cells. The humus compound is shown by the shaded parts. 6.—Starch grains from medullary ray cell. Normal grains and several grains showing how grains are now and then dissolved. The short line equals 10μ . 7.—Starch grains from diseased wood, showing how the large grains are broken up into smaller ones. (Magnification same as for fig. 6.) 8.—Transection from entirely rotted wood. The sound wood cells at one side belong to a small piece of more resistant wood. (Magnification same as for fig. 2.)

PLATE V. Cross section of diseased trunk of the white ash kept in a moist place for several weeks. The fungus hyphæ have grown out from the diseased wood, forming a white felt.



FIG. 1.—FRUITING BODY OF POLYPORUS FRAXINOPHILUS.



FIG. 2.—TWO YOUNG SPOROPHORES ON LIVING ASH.



FIG. 3.—AN OLD SPOROPHORE ON LIVING ASH.

FRUITING BODIES OF POLYPORUS FRAXINOPHILUS ON WHITE ASH.

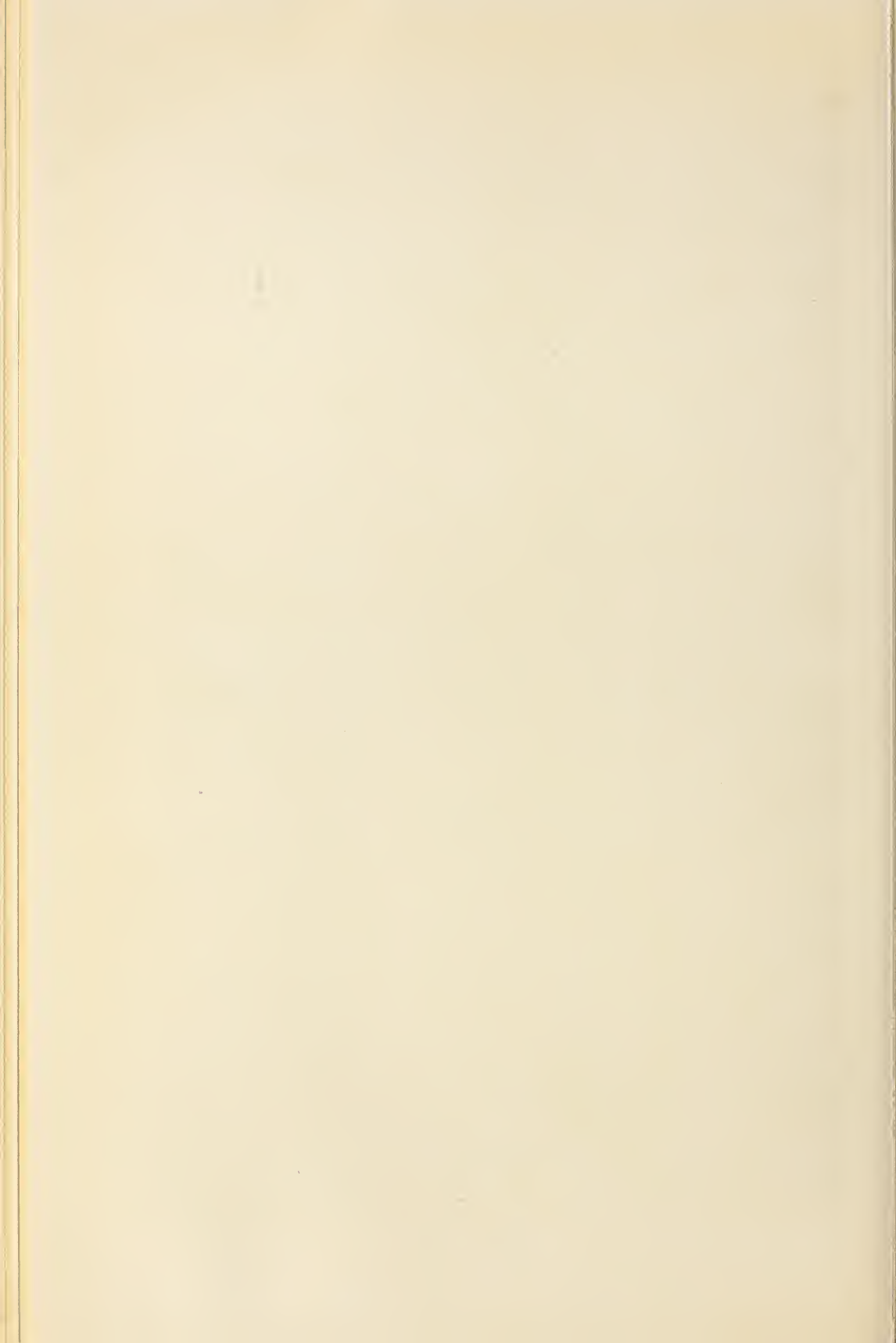


FIG. 1.—HEALTHY ASH WOOD SHOWING STARCH IN THE MEDULLARY RAYS.

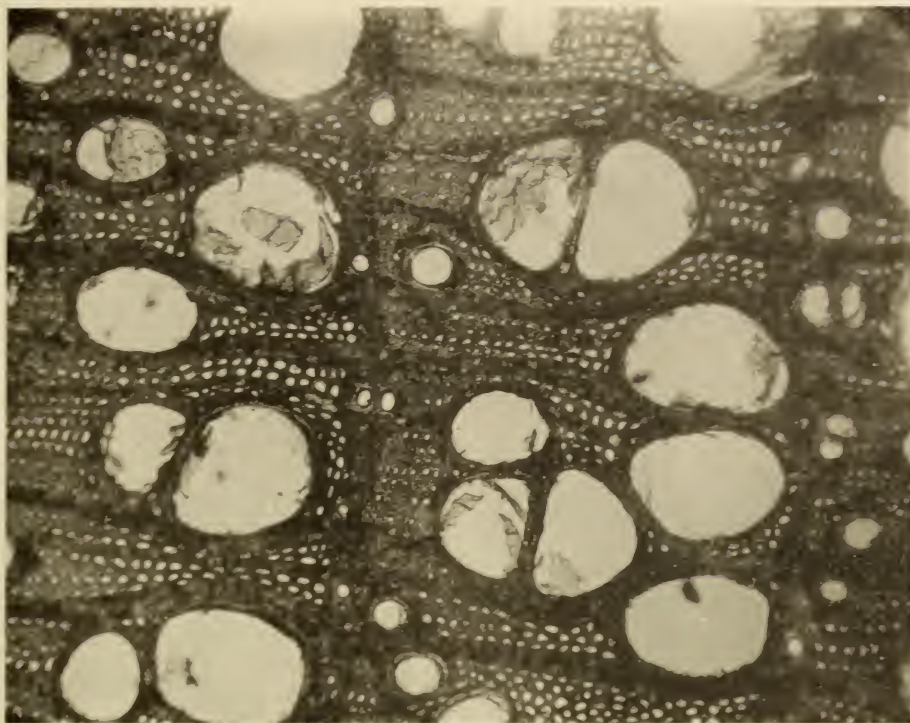
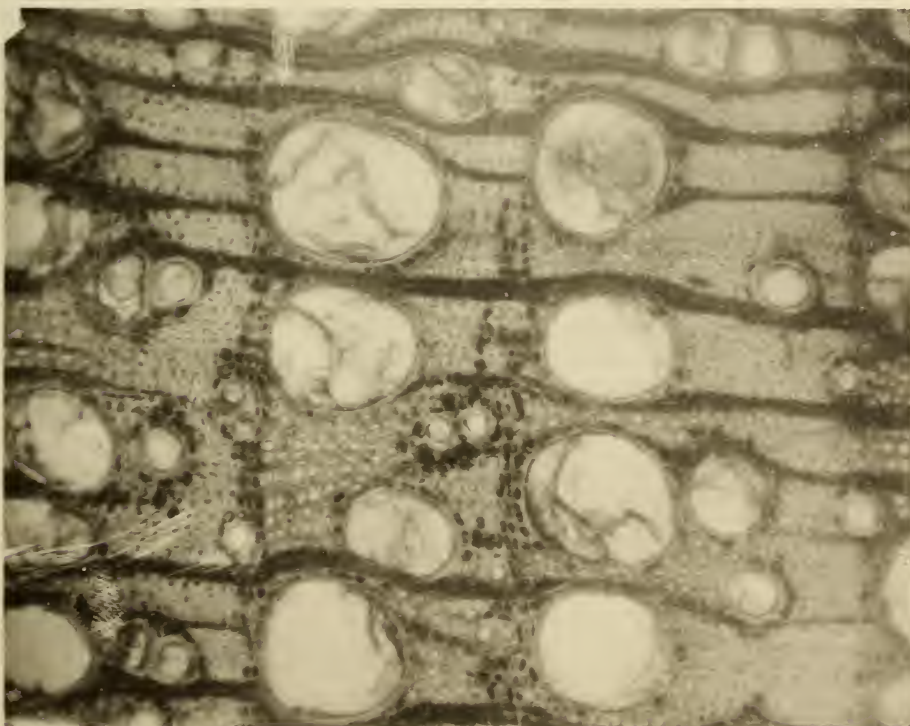
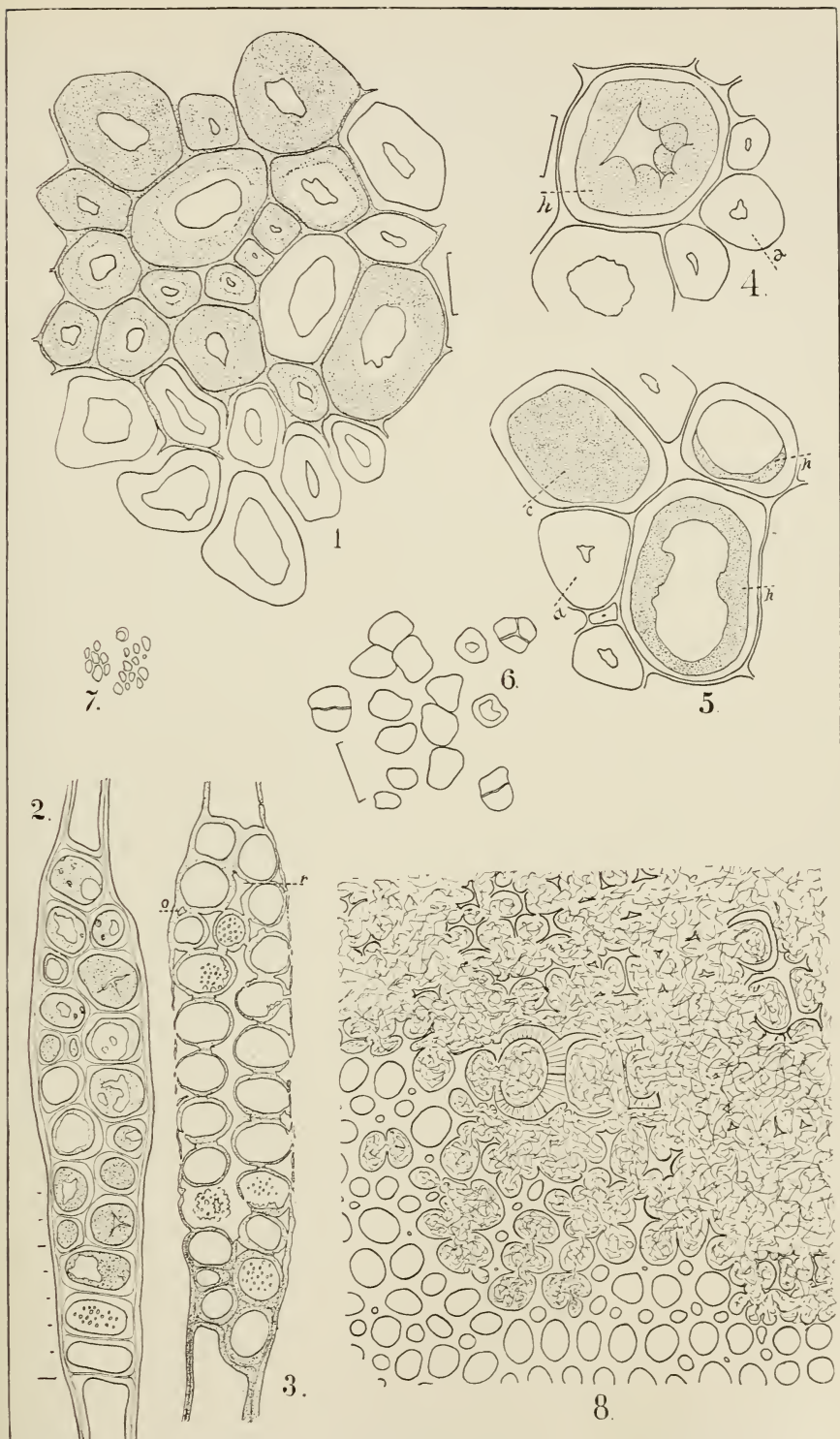
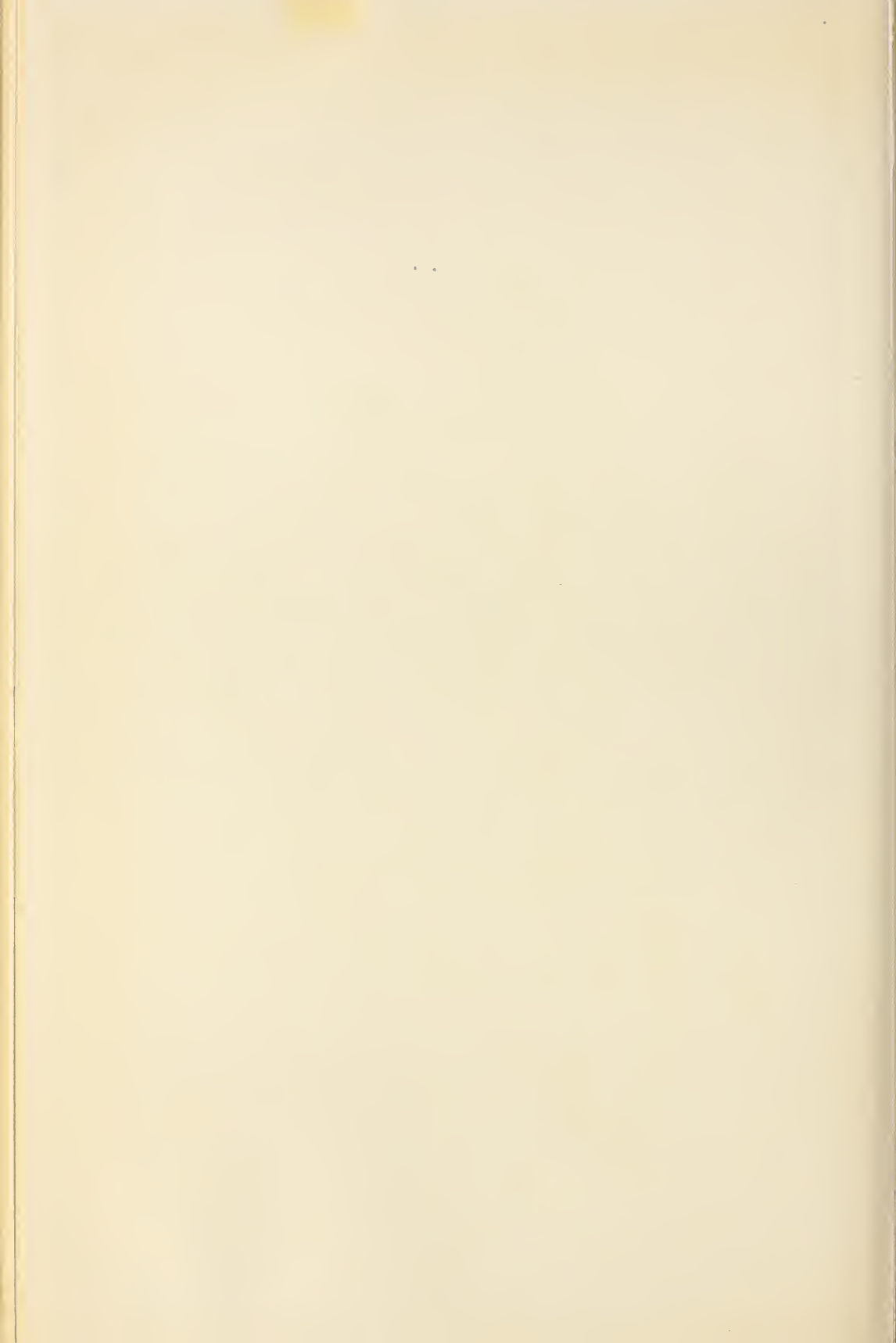


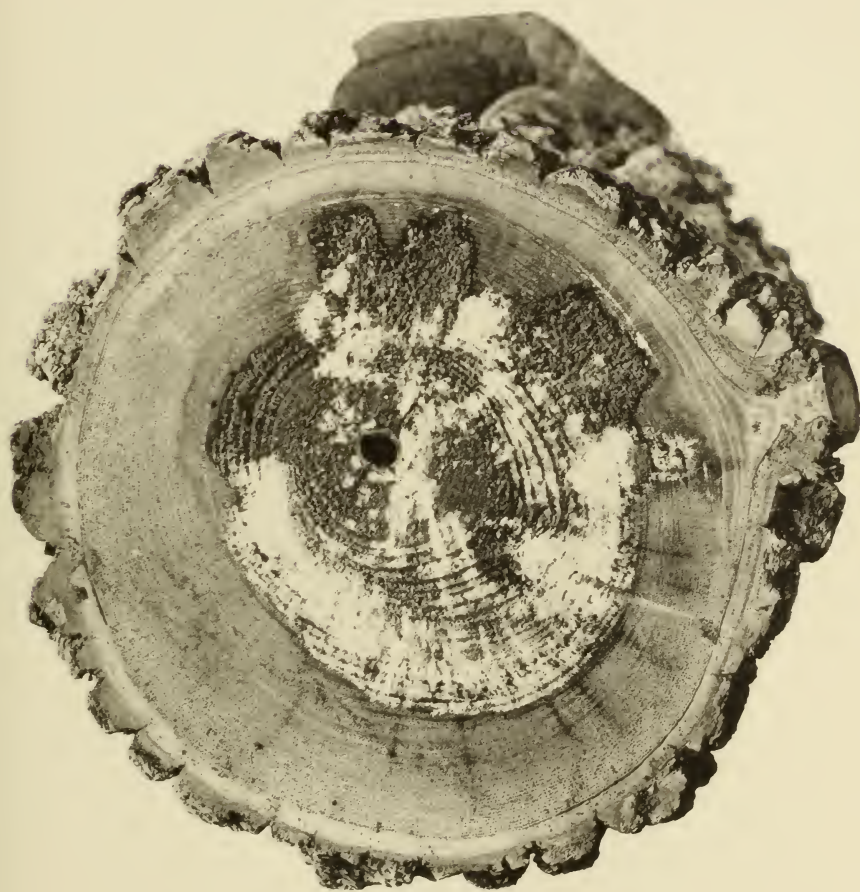
FIG. 2.—DISEASED ASH WOOD SHOWING HUMUS COMPOUND



DISEASE CAUSED BY *POLYPORUS FRAXINOPHILUS*.

- 1, Transection of ash wood; 2, transection of medullary ray; 3, medullary ray, showing later stage of fungus attack; 4, 5, transection of wood cells; 6, starch grains from medullary ray cell; 7, starch grains from diseased wood; 8, transection from entirely rotted wood.





CROSS SECTION OF DISEASED TRUNK OF WHITE ASH KEPT IN A MOIST PLACE FOR SEVERAL WEEKS
(SHOWING GROWTH OF MYCELIUM FROM THE ROTTED PART.)

